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INSIDE

Genetic predisposition
of alcoholism and more

Editorial

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“The greatest medicine of all is to teach people how not to need it.”
– Hippocrates (attrib.)

Greetings Readers! I would like to take this opportunity to introduce you to the **Journal of Public Health and Allied Sciences (JPHAS)**. An official publication of **Cephorb Medline International Pvt. Ltd.**

In times when modern medicine is advancing by leaps and bounds, we have technology and knowledge to treat and limit many life threatening conditions. We are running side by side with the diseases in hope to catch up with them and in best case – overtake them.

Public health is the science and art of preventing the disease. We attempt to prevent the disease even before it shows its first signs and symptoms. If we need to treat a preventable disease, then it is an indicator that we failed at our primary objectives altogether.

The aim of this journal is to promote and disseminate public health research to an audience at the global level. The scope of this journal, like Public Health itself, is multi-disciplinary. We invite research from Medicine, Dentistry, Nursing, Pharmacy and other Allied Health Sciences. Studies from the fields benefitting Public Health are also invited viz. Social Sciences, Environmental Engineering, and Anthropology etc.

This issue features insightful studies done on Genetic predisposition of alcoholism, Child abuse, Dentures as a nidus for opportunistic infections and more. I hope the readers enjoy browsing this issue and provide constructive feedback on the same. I would also like to thank the Co-founders and Editorial Board of JPHAS for making this dream, a reality.

Happy Reading!

Dentures as a Nidus for an Opportunist: A Comparative Study

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Abstract

Introduction: The oral flora changes with loss of teeth in the oral cavity and candida albicans is the most opportunist microorganism associated with candida-associated denture stomatitis. In current situation the different factors which are advocated in denture induced stomatitis includes immunity, diabetes and other diseases. So, overall wellbeing of patient should be evaluated.

Purpose: The aim of study was to determine the fungal occurrence rate in the oral cavity of denture wearer patients in comparison to those without dentures.

Material and Methods: The study was conducted in patients who were treated in a dental college. Evaluation concerning dentition and oral hygiene was performed. Samples for mycological examinations were taken from palatal mucosa, the tongue surfaces and mucosal surfaces of patients with dentures while tongue and palate swabs were taken from those without dentures. For culture and identification of fungi standard methods were used.

Result: Dental and mycological examinations were performed in 24 patients, out of which 14 (58.3%) used complete or partial dentures and 10 (41.7%) had their own dentition (without dentures). Oral cavity revealed only growth of *Candida albicans* species, more frequently in patients with dentures (9/14; 64.3%) than in those without dentures (3/10; 30%) ($p=0.0003$). In 9 (64.3%) denture wearer

patients, denture stomatitis was observed, associated mainly with *C. albicans* infections.

Conclusion: The occurrence rate of oral *Candida albicans* in patients with dentures was higher than in patients without dentures ($p<0.05$).

Key words: oral *Candida albicans*, denture plaque, denture wearers, denture stomatitis, oral hygiene.

Introduction

Candida infections receive increasing attention, presumably due to the increased prevalence worldwide. Several studies have shown that several Candida species possess a multitude of virulence mechanisms leading to flourishing colonization and infection of the host when appropriate conditions occur. [1] Davenport results show that candidal carriage is more in males than females [2]. The acknowledgment that Candida is a significant pathogen that has led to many laboratory studies evaluating this virulence attributes in an attempt to elucidate the pathogenesis of the disease. The various advancements are made in understanding some of these features, such as the mechanisms that result in adherence to surfaces, cell colonize various habitats in humans, notably oral mucosa and skin. Commensal existence of oral Candida species varies from 20% to 50% in a healthy dentulous population and Denture wearing is a predisposing factor for oral colonization by candida and its prevalence can increase from 60 to 100%

growth on surfaces is a natural part of the *Candida* lifestyle, one can expect that *Candida* colonizes denture. [3]

There is a large body of evidence indicating that *Candida* is able to adhere to acrylic resin dentures. This is the first step that may lead to the development of the infectious process and that may ultimately result in varying degrees of denture stomatitis of the adjacent mucosa. *Candida* adheres directly or via a layer of denture plaque to denture base (polymethylmethacrylate – PMMA). [4] Without this adherence, micro-organisms would be removed from the oral cavity when saliva or food is being swallowed. It is well-known that numerous factors are involved in the adhesion of *Candida* to the acrylic resin base, surface free energy, substrate surface properties, as surface charge, hydrophobicity, and irregularity have all been advocated to influence the initial adhesion of microorganisms. Microbial adhesion on biomaterial surfaces depends on composition of biomaterials and surface structure and on the physicochemical properties of the microbial cell surface, again its surface charge and hydrophobicity. Components of the resilient denture liners and acrylic resin may reduce the adhesion and inhibit the growth of *Candida*. [1]

Material and Methods

A total of 24 patients were evaluated in this study who were wearing dentures and without dentures were selected by purposive non-probability sampling method. Each patient completed a medical and dental history and signed an informed consent document. All patients accepted oral examinations. The patients were divided into two groups, according to the presence or absence of dental prosthesis (denture). Samples were collected with swab from oral mucosa (palatal mucosa and tongue dorsa) of all patients and the adjacent denture surfaces of patients with dental prosthesis. All oral specimens were placed on Sabouraud glucose agar. All isolated yeasts were identified with classic methods.

Statistical analysis

Data was analysed on computer program SPSS version 13. Student's t-test was used to analyse the differences between the means (shown as mean \pm s.d.). The Chi-squared test was used to analyse the differences between the frequencies in groups. Groups or subgroups were considered significantly different from each other if $P < 0.05$.

Results

The prospective studies were performed in 24 patients treated in the dental college, out of which, 14 (58.3%) used complete or partial dentures and

10 (41.7%) had their own dentition (patients without denture).

A total of 12 (50.0%) candidal strains from examined patients were isolated. In 9/12 (75.0%) patients *Candida albicans* strains were recovered from both in palatal mucosa and tongue dorsa. Oral cavity revealed only yeasts of *Candida albicans* species, more frequently in patients with dentures (10/14; 71.4%) than in those without dentures (2/10; 20.0%) ($p = 0.0003$).

Among putative risk factors evaluated, wearing denture patients and only older age (55-74 years: 7/10; 70.0% vs <44 years old: 1/4; 25.0%) ($p = 0.01$) were associated with the most frequent isolated *C. albicans*. We also demonstrated that the presence of *C. albicans* was more frequent in denture-related stomatitis (6/9; 66.7% +ve vs 4/5; 80.0% -ve) ($p = 0.0107$). The presence of denture-related stomatitis was assessed according to a modified version of Newton's classification. The severity of the palatal inflammation was classified according to Newton's types no stomatitis, no evidence of palatal inflammation; stomatitis Newton type I, II and III respectively, petechiae dispersed throughout all or any part of palatal mucosa in contact with the denture; macular erythema without hyperplasia; and diffuse or generalized erythema with papillary hyperplasia.

Statistical analysis showed that the frequency and the category of stomatitis between the risk factors was not significantly different ($p > 0.05$), except for tongue disorders. Frequency of *C. albicans* present in patients with stomatitis and tongue disorders (mainly atrophic tongue) was significantly higher than in free-stomatitis patients with tongue disorders. Statistical analysis according to Newton types had shown no significant relation between stomatitis and number of detected yeast colonies (density as CFU) on dentures (denture plaque) and palatal mucosa and/or tongue dorsa.

Discussion

Candidal infections are a major problem in the world, especially among the immunosuppressed people. The ability of *Candida* to pass through the tissues is the first step of the infectious process. It has been seen that the forms capable with hyphae are able to stick and to invade more quickly the tissues of the host. [5] The microorganisms present in the oral cavity interact between them in various ways, as using directly their own metabolic products or exchanging themselves molecular signals. Several studies have shown that the co-aggregation includes protein-carbohydrates interactions. [6,7]

Traumas are considered as the main liable to determine *Candida*-associated denture stomatitis

with none association with the microbial communities and the presence of denture. [8]

Candida colonization in denture wearers, especially immunocompromised patients, can be disruptive to dental treatment and may be a barrier to patient health. The surface irregularities of acrylic resin are a factor in the entrapment of microorganisms, especially *Candida albicans*. Consequently, controlling the spread of fungal infection in risk patients who wear removable prostheses and who are more susceptible to fungal infections because of their immunosuppression is of critical importance. It is important in denture wearers with HIV infections and diabetic patients or advanced carcinoma.

The results of our study support the hypothesis that there is a significant quantitative alteration in the candidal count after the complete denture is worn for a month.

Oral fungal infections frequently develop in individuals with advanced cancer, especially in the patients receiving palliative care for advanced malignant disease. Bagget *et al.* showed that patients with advanced cancer have demonstrated a high incidence (51%) of oral colonization with non-*C. albicans* yeasts, many of which had reduced susceptibility to fluconazole and intraconazole (e.g. 72% resistant *C. glabrata*). [9] The oral yeast carriage in 66% of patients with advanced cancer by Davies *et al.* were observed. [10] The frequency of isolation of individual species was: *Candida albicans*, 46%; *C. glabrata*, 18%; *C. dubliniensis*, 5%; others, <5%. Oral yeast carriage was associated with denture wearing ($p=0.006$). [5] In our study also a high isolation frequency (76% or 87.5% for denture wearers) was observed, but only one species, *Candida albicans*, from patients with abdominal cancer.

The results in our study also show that denture-related stomatitis (denture stomatitis), the presence (colonization/infection) of *Candida albicans* on the denture is probably linked to extensive inflammation. In addition, the isolated *Candida* spp. associated with dentures are related to the poor hygienic condition of the prostheses, to the long-time of usage, wearing dentures at night and to the modifications of the hard supporting tissues. Whether *C. albicans*, alone or with other organisms, is involved in onset or development of denture related stomatitis remain to be determined.

Conclusion

A positive association was demonstrated between dentures and candidiasis in this study, we recommend that the denture wearers should use proper oral hygiene methods and avoid ill-fitting dentures. Other local and systemic predisposing factors such as xerostomia, carbohydrate usage, and

nutrient habits should be evaluated so that the spread of fungal infection is minimized in these patients.

References

1. Bellon-Fontaine MN, Mozes N, van der Mei HC, Sjollem J, Cerf O, Rouxhet PG, Busscher HJ. A comparison of thermodynamic approaches to predict the adhesion of dairy microorganisms to solid substrata. *Cell Biophys* 1990;17:93-106.
2. Davenport JC. The oral distribution of *Candida* in denture stomatitis. *British Dental Journal*, 1970;129:151-156
3. Pires FR, Santos EB, Bonan PR, De Almeida OP, Lopes MA. Denture stomatitis and salivary *Candida* in Brazilian edentulous patients. *J Oral Rehabil* 2002; 29: 1115-19.
4. Redding S, Bhatt B, Rawls, HR, Siegel G, Scott K, Lopez-Ribot J. Inhibition of *Candida albicans* biofilm formation on denture material. *Oral Surg Oral Pathol Oral Radiol Endod* .2009;107:669-72.
5. Blankenship JR, Mitchell AP. How to build a biofilm: A fungal perspective. *Curr Opin Microbiol* 2006;9:588-94.
6. Arendorf TM, Walker DM. Denture stomatitis: A review. *J Oral Rehabil* 1987;14:217-27.
7. Avon SL, Goulet JP, Deslauriers N. Removable acrylic resin disk as a sampling system for the study of denture biofilms in vivo. *J Prosthet Dent* 2007;97:32-38.
8. Redding S, Bhatt B, Rawls, HR, Siegel G, Scott K, Lopez- Ribot J. Inhibition of *Candida albicans* biofilm formation on denture material. *Oral Surg Oral Pathol Oral Radiol Endod* 2009;107:669-72.
9. Bagg J, Sweeney MP, Lewis MA, Jackson MS, Coleman D, Al MA, Baxter W, McEndrick S, McHugh S. High prevalence of nonalbicans yeasts and detection of anti-fungal resistance in the oral flora of patients with advanced cancer. *Palliat Med*, 2003; 17: 477-81.
10. Davies AN, Brailsford S, Broadley K, Beighton D. Oral yeast carriage in patients with advanced cancer. *Oral Microbiol Immunol*, 2002; 17: 79-84.

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Genetic Predisposition to Alcoholism

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Introduction

Substance abuse is a major threat to public health worldwide, it refers to the harmful or hazardous use of psychoactive substances, including alcohol and illicit drugs. [1] Use of psychoactive substance can lead to dependence syndrome, a cluster of behavioural, cognitive, and physiological phenomena that develops after repeated substance use and that typically includes a strong desire to take the drug, difficulties in controlling its use, persisting in its use despite harmful consequences, a higher priority given to drug use than to other activities and obligations, increased tolerance, and sometimes a physical withdrawal state. [2] Among these, one of the major problems the world is facing is alcoholism. [3] The American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders DSM-V, 2013, explains that activation of the brain's reward system is central to problems arising from drug use, which essentially means the rewarding feeling that people experience as a result of taking drugs may be so profound that they neglect other normal activities in favour of taking the drug. The manual recognizes substance related disorders resulting from the use of ten separate classes of drugs: Alcohol; Caffeine; Cannabis; Hallucinogens (phencyclidine or similarly acting arylcyclohexylamines, LSD); Inhalants; Opioids; Sedatives; Hypnotics; Anxiolytics; Stimulants (including amphetamine-type substances, cocaine and other stimulants); Tobacco; Other or Unknown substances. [4]

Alcoholism

The World Health Organization (WHO) defines alcoholism or alcohol dependence as "chronic and continual drinking or periodic consumption of alcohol characterized by impaired control over drinking, frequent episodes of intoxication and pre-occupation with alcohol and use of alcohol despite adverse consequences". In narrower concept as a

"disease marked by loss of control over drinking caused by pre-existing biological abnormality and having a predictable progressive course". [5] Excessive consumption of alcohol is a leading public health concern and is a massive global health burden. It is estimated that worldwide 3.3 million deaths every year result from harmful use of alcohol, this represents 5.9 % of all deaths. [3] Alcohol consumption is a causal factor in more than 200 disease and injury conditions. It is associated with a risk of developing health problems such as mental and behavioural disorders, including alcohol dependence, major non-communicable diseases such as liver cirrhosis, some cancers and cardiovascular diseases, as well as injuries resulting from violence and road clashes and collisions. The latest causal relationships are those between harmful drinking and incidence of infectious diseases such as tuberculosis as well as the course of HIV/AIDS. Alcohol consumption by an expectant mother may cause foetal alcohol syndrome and pre-term birth complications. [6]

Magnitude of problem in India

According to the WHO report, in the year 2010 about 30% of the entire population of India consumed alcohol. 93% of alcohol was consumed in the form of spirits, followed by beer with 7% and less than 1% of the population consumed wine. [7] The per capita consumption of alcohol in the country increased from 1.6 litres in 2003-2005, to 2.2 litres in 2010-2012. An average individual over the age of 15 consumed over 8 litres of alcohol per annum in the South Indian states followed by Maharashtra and Punjab. Over 11% of the population in India indulged in heavy or binge-drinking. On the 'Years of Life Lost' scale, which is based on alcohol-attributable years of life lost, India has been rated 4 on a scale of 1 to 5. This implies that the alcohol consuming population of

our country loses most years of their life because of drinking and its consequences. [7] Alcoholism is a chronic, multifactorial psychiatric disorder which is determined by numerous physiological, genetic, psychosocial and environmental aspects thus the advancement of alcoholism is complicated among all of the underlying factors genetic and environmental factors are difficult to control. Not all individuals who consume alcohol become alcoholics, one of the triggers is the vulnerability or biological susceptibility, which is high in alcoholism. [8] Biological susceptibility point towards all the conditions that an individual is born with, that make an individual more or less vulnerable to developing a pathological condition. An understanding of susceptibility studies at biological and especially genetic level is relatively less. On the contrary understanding of these aspects should be given high priority as this would result in the crucial advances in our ability to treat and prevent drug addiction. As for instance, the discovery of a specific genetic effect on the development of alcoholism would be beneficial for the reasons that it would lead to the identification of some people at risk, who could act to avoid developing alcohol-related problems, it may help us to understand the role of environmental factors that are critical in the development of alcoholism and it may lead to better treatments, based on new understandings of the physiological mechanisms of alcoholism. [9]

Genetic vulnerability to Alcoholism

Heritability studies propose that an individual's genotype imparts a particular level of susceptibility, or risk. Various comparative studies across populations have recommended that sociocultural factors determine differences in thresholds above which an individual is likely to go past social drinking and slip into abuse or addiction. The progression of alcoholism is due to a distinctive set of biochemical and neurobiological determinants or are the causes of addiction common to many substances. [10]

Researchers have examined possible genetic components of alcoholism by studying populations and families as well as genetic, biochemical, and neurobehavioral markers and characteristics. Two major methods of investigating the inheritance of alcoholism are studies of twins and of adoptees [9]. Twin studies compare the incidence of alcoholism in identical twins with the incidence of alcoholism in fraternal twins. The presence of genetic component will be confirmed if identical twins exhibit similar histories of developing alcoholism or not developing alcoholism. Additionally, it was found that fraternal twins differ in their propensities to develop alcoholism which strengthened the study further. [11,12] The researchers found greater concordance of alcohol

dependence in identical twins than in fraternal twins. In adoption studies there is comparison in the histories of children of alcoholics who are adopted by non-alcoholics and grow up in a nondrinking environment with the histories of children of non-alcoholics similarly raised in a nondrinking environment takes place. If genetic factors are involved, the preferential development of alcoholism in adulthood takes place. The expected trends were evident in a pioneer study of adopted Danish children. In another much larger studies of adoptees, also revealed same trends in which it was hypothesized that children of alcoholics characterized as having an early onset of drinking problems, usually being male, and displaying personality disorders such as antisocial behaviour had a more heritable form of alcoholism. [11,12] However, the argument put forward by other researchers have debated that the scenario of inheritance is more complicated, and what is inherited is a combination of personality traits, such as those associated with antisocial behaviour, rather than alcoholism itself. [13] Different models have been suggested for the way in which alcoholism runs in families, by a restricted number of family studies. Interpretation of these studies has been complicated by the probability that alcoholism is a heterogeneous condition, that is, a collection of different conditions that look similar, but whose mechanisms and modes of inheritance may differ. Additional studies are needed to sort out the mechanisms of transmission. [14] Population and family studies cited above tries on establishing the presence of a broad genetic impact on alcoholism. Genetic marker studies have been employed by the researchers for the investigation of specific genes. On the assumption, that if specific human genes are related to alcoholism, then genes lying close to them on the same chromosome--and the traits they determine may be inherited at the same time the risk of alcoholism is inherited. [15]

Specific Gene Candidates

Genes might play a direct role in the development of alcoholism, by affecting the body's alcohol metabolism; or they may possibly play a less direct role, influencing a person's temperament or personality in such a way that the person becomes vulnerable to alcoholism. Alcohol's genetic susceptibility may have been instigated in personality traits by means of anxiety, dysphoria and impulsivity, which predisposes to alcohol seeking behaviour. Behavioural disorders may increase the risk in alcohol abuse. [9]

Genes Encoding Proteins Involved in Alcohol Metabolism

The genes linked with alcohol dependence are most frequently are those encoding the enzymes involved in alcohol metabolism. [16] The principal

pathway of alcohol metabolism comprises of two steps. The first step involves conversion of ethanol into the toxic intermediate acetaldehyde; facilitated by the alcohol dehydrogenase (ADH) enzymes. In a second step, the acetaldehyde splits into acetate and water by aldehyde dehydrogenase (ALDH) enzymes. [17]

Several variants (i.e., alleles) of the genes encoding the ADH and ALDH enzymes exist, leading to variations (i.e., polymorphisms) in the sequence of the DNA building blocks. A significant group of ADH enzymes are the ADH class I isozymes; ADH1A, ADH1B, and ADH1C. For both the genes encoding ADH1B and those encoding ADH1C, altered proteins products of several alleles have been recognized, and the proteins encoded by some of these alleles demonstrate high enzymatic activity in laboratory experiments (i.e., in vitro). This suggests that in people carrying these alleles, ethanol is more rapidly converted to acetaldehyde. [18] Additionally, genome-wide screening has been done to recognize genes associated with alcoholism and alcohol-related traits. Three independent trials comprising largely of population of European origin—the COGA study [19], the Irish Affected Sib Pair Study of Alcohol Dependence [20], and an Australian sample. [21] These studies have found evidence that a region on chromosome 4 containing the ADH gene cluster shows linkage to the phenotypes studied. This cluster contains several other polymorphic genes which have been associated with alcohol dependence in addition to the genes encoding ADH class I isozymes. [22]

Genes Encoding Proteins Involved in Neurotransmission

Activities of numerous neurotransmitter system is influenced by alcohol, example γ -aminobutyric acid (GABA), dopamine, and acetylcholine, and naturally occurring compounds which structurally resemble opioids and cannabinoids. Hence, certain genes encoding elements of these neurotransmitter systems may heighten the risk of alcohol dependence. [23]

GABA_A Receptor

GABA is engaged in numerous behavioural effects of alcohol, including motor incoordination, anxiety reduction (i.e., anxiolysis), sedation, withdrawal symptoms, and inclination for alcohol. GABA_A receptor genes on chromosome 4 were constantly recognized in genome-wide scans observing for genes associated with alcohol dependence. Thus, COGA analysis methodically examined small DNA segments of well-known location (i.e., genetic markers), situated in the GABA_A receptor gene cluster on chromosome 4. Substantial association between GABRA2 gene and alcohol dependence was established in these investigations. [24]

Thus, different forms of GABA_A receptor gene are associated with alcohol dependence, alcohol-related phenotypes (e.g., history of alcohol-induced blackouts and age at first drunkenness), and other aspects of drug dependence. [25]

Cholinergic System

The cholinergic system includes neurons that either release the neurotransmitter acetylcholine or respond to it and is involved in processes as arousal, reward, learning, and short-term memory. in the COGA sample, linkage was observed between the gene responsible for acetylcholine and alcohol dependence, also subsequent experiments confirmed that an association existed between alcohol dependence and the Acetylcholine gene. [26]

Additional investigation in the COGA sample have proposed that Acetylcholine gene is linked with an amplified risk of externalizing conditions, as well as symptoms of alcohol dependence and drug dependence. [27]

Genes Involved in the Endogenous Opioid System

Endogenous opioids are small molecules naturally produced in the body that have similar effects as the opiates (e.g., morphine and heroin) and which, among other functions, modulate the actions of other neurotransmitters. ZZ, opiates, and cocaine. This is supported by the finding that the medication naltrexone, which prevents the normal actions of endogenous opioids (i.e., is an opioid antagonist), is useful in the treatment of alcohol dependence and can reduce the number of drinking days, amount of alcohol consumed, and risk of relapse. [28]

main gene candidate is OPRM1, which encodes one type of opioid receptor (i.e., the μ -opioid receptor), although the results so far have been equivocal. This gene contains a polymorphism resulting in a different protein product (i.e., a non-synonymous polymorphism) that in one study was found to bind one of the endogenous opioids (i.e., β -endorphin) three times as strongly as the main variant of the gene; other studies, however, could not confirm this finding. Laboratory studies have suggested that OPRM1 is associated with sensitivity to the effects of alcohol. [16]

Endogenous Opioid System

Endogenous opioids are small molecules naturally produced in the body that have similar effects as the opiates (e.g., morphine and heroin) and responsible for the modulation of actions of other neurotransmitters. ZZ, opiates, and cocaine. The role of endogenous opioid system in addiction is sustained by the finding that prescription drug Naltrexone, for the treatment of alcohol dependence is an antagonist to endogenous opioid

This drug can decrease the number of drinking days, quantity of alcohol consumed, and threat of relapse. [28]

The main gene candidate is opioid receptor mu 1, although the results so far have been unclear. Polymorphism in this gene results in an altered protein product that binds to one of the endogenous opioids- β -endorphin three times as strongly in comparison to wild type gene; although except one other studies failed to replicate the association. Laboratory studies have suggested that opioid receptor mu 1 is associated with sensitivity to the effects of alcohol. [16, 29, 30]

Endogenous Cannabinoid System

Endogenous cannabinoids are naturally producing compounds, structurally analogous to the psychoactive compounds found in the cannabis plant and which binds to cannabinoid receptors. The function of endogenous cannabinoid system is to control brain circuits through dopamine neurotransmitter, which possibly assist in mediating the rewarding experiences related with addictive substances. [31]

Cannabinoid Receptor Type 1 is the principal cannabinoid receptor in the brain. The research findings to date regarding the association between cannabinoid receptor type 1 gene and Alcohol dependence have been ambiguous, as some studies generating positive results [32] whereas other generated negative results. [33] A recent case-control study found that numerous genetic variants in Cannabinoid Receptor 1 gene were significantly associated with alcohol dependence. [34]

Comorbidity of Alcohol

Alcohol, cocaine, opiate, and tobacco (nicotine) dependency co-occur more often in the population than would be expected from their frequencies. This raises the possibility that there may be substance-general, as well as substance-specific, components to the heritability of alcoholism. [35]

Both alcoholism and drug disorders are familial; two large studies have evaluated the familial aggregation of alcohol and drug dependence. [36] Both studies found that relatives of drug-disorder probands across a wide range of substances, including opioids, cocaine, and cannabis, had a greater rate of drug disorders themselves than relatives of controls. [37] However, this comorbidity occurred largely independently from co-transmission of alcoholism, suggesting that the transmission of alcoholism and other drug disorders is largely independent.

The strongest evidence of a shared, as well as a specific, addictive tendency is between alcohol and nicotine. It has long been observed that there is a relationship between smoking and alcoholism. More than 80% of alcoholics smoke cigarettes and 70% are heavy smokers, compared with 30% of the

general population who smoke and 10% who smoke heavily. [38]

Epigenetic aspects of alcoholism

Monozygotic twins' differing response to alcohol establishes that environmental and epigenetic factors are significant in predisposition to alcohol abuse. Additionally, diverse and contradictory results of genetic polymorphisms related to alcoholism studies point out that in addition to the alleles, multifactorial inheritance, genetic heterogeneity, the differences in response are also due to DNA (gene) alterations at an epigenetic level example at its expression (transcription and translation) or its regulation. [40]

There are different epigenetic mechanisms such as DNA methylation, the ADP-ribosylation, sumoylation, acetylation, methylation, phosphorylation, deamination, isomerization, ubiquitination of histones, non-coding RNAs, among others. Gene-environment interactions can be facilitated to epigenetic mechanisms and evidence is emerging that alcohol consumption is one environmental factor which can alter epigenetic signatures and thus related gene expression levels. Altered gene expression in brain reward regions after alcohol intake has been reported, which suggests that individual genes are differentially regulated following alcohol consumption. [41]

Studies in humans and laboratory animals have demonstrated that both chronic and acute alcohol consumption affects many aspects of the circadian cycle and physiological functions, endocrine and behavioural. [42]

Conclusion

Substantial amount of evidences advocate that genetic factors influence the risk of development of alcoholism, with heritability estimating 50 percent and higher. Twin and Adoptee studies indicate the risk of developing alcohol dependence through inheritance. These interpretations propose that person's liability for alcohol dependence will result partly from a general externalizing factor and partly from genetic factors that are more disorder-specific. Although, there is good evidence for substantial heritability for alcoholism, individual differences in clinical presentation suggest variation in origins of vulnerability.

Alcoholics vary in their drinking patterns, the severity of their symptoms, and in behavioural, physical, and psychiatric sequelae. Vulnerability may reside in personality or psychiatric traits that predispose to alcohol-seeking behaviour, differential response to the effects of alcohol, or differential predisposition to addiction.

Further research is needed to determine the significance of genetic anomalies in alcoholism. The genetic defects implicated might be useful in

designing Prevention and treatment regimens for alcohol-related disorders.

References

1. WHOint. (2016). *World Health Organization*. Retrieved 25 May, 2016, from http://www.who.int/topics/substance_abuse/en/
2. WHOint. (2016). *World Health Organization*. Retrieved 25 May, 2016, from http://www.who.int/medicines/areas/quality_safety/6.1information.pdf
3. World Health Organization. 2014. *Global Status Report on Alcohol and Health-2014ed*. Retrieved 25 May, 2016, from http://www.who.int/substance_abuse/publications/global_alcohol_report/msb_gsr_2014_1.pdf
4. Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition. May 2013
5. WHOint. (2016). *Who.int*. Retrieved 25 May, 2016, from http://www.who.int/substance_abuse/terminology/who_lexicon/en/
6. Shield, K.D. , Parry, C & Rehm, J. (2013). Chronic Diseases and Conditions Related to Alcohol Use. *Alcohol Research: Current Reviews*, 35(2), 155-173.
7. World Health Organization. (2016). 2014 Global Alcohol Report. Retrieved 25 May, 2016, from http://www.who.int/substance_abuse/publications/global_alcohol_report/profiles/ind.pdf
8. NIHgov. (2016). *Nihgov*. Retrieved 25 May, 2016, from <http://pubs.niaaa.nih.gov/publications/arh31/14-25.htm>
9. NIHgov. (2016). *Nihgov*. Retrieved 25 May, 2016, from <http://pubs.niaaa.nih.gov/publications/aa18.htm>
10. Enoch & goldman. (2002). Molecular and Cellular Genetics of Alcohol Addiction. In Davis et al (Eds), *Neuropsychopharmacology: The Fifth Generation of Progress* (pp.1413-1424). Philadelphia: Lippincott, Williams, & Wilkins.
11. Heath, A.C. (1995). Genetic Influences on Alcoholism Risk . *Alcohol Health & Research World*, 19(3), 166-171.
12. Verhulst, B, Neale, M.C & Kendler, K.S. (2015). The heritability of alcohol use disorders: a meta-analysis of twin and adoption studies. *Psychological Medicine*, 45(5), 1061-1072.
13. Peele, S. (1986). The Implications and Limitations of Genetic Models of Alcoholism and Other Addictions. *Journal of Studies on Alcohol*, 47(1), 63-73.
14. Sloan, C.D, Sayarath, V & Moore, J.H. (2008). Systems Genetics of Alcoholism. *Alcohol Research & Health*, 31(1), 14-25.
15. National Institute of Drug Abuse. (1986). Genetic and Biological Markers in Drug Abuse and Alcoholism. Retrieved 25 May, 2016, from <https://archives.drugabuse.gov/pdf/monographs/66.pdf>
16. Dick, D & Agarwal, A. (2008). The Genetics of Alcohol and Other Drug Dependence. *Alcohol Research & Health*, 31(2), 111-118.
17. NIAAA. (2007). Alcohol Metabolism: An Update. *Alcohol Alert*, 72(1),
18. Chen et al. (1999). Interaction between the functional polymorphisms of the alcohol-metabolism genes in protection against alcoholism. *American Journal of Human Genetics*, 65(3), 795-807.
19. Saccone et al. (2000). A genome screen of maximum number of drinks as an alcoholism phenotype. *American Journal of Human Genetics*, 96(5), 632-637.
20. Prescott et al. (2005). The Irish Affected Sib Pair Study of Alcohol Dependence: study methodology and validation of diagnosis by interview and family history. *Alcoholism: Clinical and Experimental Research*, 29(3), 417-429.
21. Birley et al. (2005). Genetic Time-series Analysis Identifies a Major QTL for in vivo Alcohol Metabolism Not Predicted by in vitro Studies of Structural Protein Polymorphism at the ADH1B or ADH1C Loci. *Behavior Genetics*, 35(5), 509-524.
22. Zakhari, S. (2013). Alcohol Metabolism and Epigenetics Changes. *Alcohol Research: Current Reviews*, 35(1), 6-16.
23. Herz, A. (1997). Endogenous opioid systems and alcohol addiction. *Psychopharmacology*, 129(1), 99-111.
24. Xuei et al. (2006). Association of the kappa-opioid System with alcohol dependence. *Molecular Psychiatry*, 11(11), 1016-1024.
25. Dick et al. (2006). Association Between GABRA1 and Drinking Behaviors in the Collaborative Study on the Genetics of Alcoholism Sample. *Alcoholism: Clinical*

- and Experimental Research, 30(7), 1101-1110.
26. Luo et al. (2005). CHRM2 gene predisposes to alcohol dependence, drug dependence and affective disorders: results from an extended case-control structured association study. *Human Molecular Genetics*, 14(16), 2421-2434.
 27. Hendershot et al. (2011). Preliminary Evidence for Associations of CHRM2 with Substance Use and Disinhibition in Adolescence. *Journal of Abnormal Child Psychology*, 39(5), 671-681.
 28. Thorsell, A. (2013). The μ -Opioid Receptor and Treatment Response to Naltrexone. *Alcohol and Alcoholism*, 48(4), 402-408.
 29. Bond et al. (1998). Single-nucleotide polymorphism in the human mu opioid receptor gene alters β -endorphin binding and activity: Possible implications for opiate addiction. *Proceedings of the National Academy of Sciences of the United States of America*, 95(16), 9608-9613.
 30. Valenta et al. (2013). μ -Opioid receptors in the stimulation of mesolimbic dopamine activity by ethanol and morphine in Long-Evans rats: a delayed effect of ethanol. *Psychopharmacology*, 228(3), 389-400.
 31. Fonseca et al. (2004). THE ENDOCANNABINOID SYSTEM: PHYSIOLOGY AND PHARMACOLOGY. *Alcohol and Alcoholism*, 40(1), 2-14.
 32. Zhang et al. (2004). Human cannabinoid receptor 1: 5' exons, candidate regulatory regions, polymorphisms, haplotypes and association with polysubstance abuse. *Molecular Psychiatry*, 9(10), 916-931.
 33. Herman et al. (2006). Association study of the CNR1 gene exon 3 alternative promoter region polymorphisms and substance dependence. *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics*, 141B (5), 499-503.
 34. Zuo et al. (2007). CNR1 variation modulates risk for drug and alcohol dependence. *Biological Psychiatry*, 62(6), 616-626.
 35. Tsuang et al. (1998). Co-occurrence of abuse of different drugs in men: the role of drug-specific and shared vulnerabilities. *Archives of General Psychiatry*, 55(11), 967-972.
 36. Bierut et al. (2002). Familial transmission of substance dependence: alcohol, marijuana, cocaine, and habitual smoking: a report from the Collaborative Study on the Genetics of Alcoholism. *Archives of General Psychiatry*, 59(2), 982-988.
 37. Merikangas et al. (2009). The familial aggregation of cannabis uses disorders. *Addiction*, 104(4), 622-629.
 38. NIAAA. (1998) Alcohol and tobacco. In: *Alcohol alert*. Washington, DC: NIAAA;39:1-4.
 39. Farrell et al. (2001). Nicotine, alcohol and drug dependence and psychiatric comorbidity. *The British Journal of Psychiatry*, 179(5).
 40. Foroud et al. (2010). Who Is At Risk for Alcoholism?. *Alcohol Research & Health*, 33(1&2), 64-75.
 41. Nieratschker, V, Batra, A & Fallgatter, A.J. (2013). Genetics and epigenetics of alcohol dependence. *Journal of Molecular Psychiatry*, 11(1).
 42. Rosenwasser, A.M. (2001). Alcohol, antidepressants, and circadian rhythms: human and animal models. *Alcohol Research & Health*, 25(1), 126-135.

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Review Article

Cleft Surgeon and Speech Pathologist – The Dream Team in their race against time in patients with Cleft Palate

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Abstract

Objective: (1) To evaluate the role of speech therapy in cleft palate patients. (2) To discuss Early vs Late surgical rehabilitation in cleft palate patients. (3) To impress the importance of a speech pathologist in the multi-disciplinary cleft team.

Design: Various databases, including MEDLINE and EMBASE, were searched between 1973 and 2015 to identify published articles relating to speech and language therapy for children with cleft palate. An exhaustive search was conducted and four reviewers independently completed inclusion assessment, data extraction, and risk of bias assessment for all studies identified.

Results: A total of 28 papers were evaluated and studies varied widely on risk of bias, intervention used, and outcome measures reported. Outcomes measures did not support either approach over the other, and based on data reported it was difficult to ascertain which approach is more effective for children with cleft palate.

Conclusion: The review found little evidence to support any specific intervention. However, our study supports the pivotal role of speech pathologist in association with a cleft surgeon in planning an ideal approach for patients with a cleft palate depending upon each type of defect. Thus, a speech pathologist and surgeon are an innate part of a team responsible in achieving ultimate functional and aesthetic harmony.

Introduction

Best results in management of cleft lip palate or other facial congenital deformities were always attained through a multidisciplinary approach. [1] Comparing to normal children it is noted that the social and communicative participation is less in cleft palate children. Because of the reduced social skills comparing to the normal peers the cleft children face problems due to the less social acceptance. This stigma later emerges in forms of negative attitude and an overall decreased acceptability of cleft children among contemporaries. [2] However, the necessity of optimal interaction with the surgeon and speech pathologists is neglected most of the times. The speech pathologist plays a major role in a cleft baby's life from the very inception right up to adulthood. The changes a speech pathologist can bring about in an operated cleft patient is noteworthy and acts as an added advantage in enhancing the surgical result as well. [3]

This review of literature thereby highlights not only the implications of cleft palate on speech and language but also stresses upon the importance of interaction between the speech pathologist and the cleft surgeon in attaining the best possible results of a cleft surgery.

Materials and Method

Several electronic databases were searched include MEDLINE. A hand search was performed among reference lists and relevant journals

Discussion

Disorders associated with cleft palate

Cleft babies are plagued by multiple problems such as developmental delays, feeding difficulties, abnormal speech, hearing loss and most importantly - the psychosocial issues related to stigma due to various communication disorders. [1] Communication is the ability to interact by speech (Table1). Difficulty in communication stems from several factors like cleft type/severity, associated syndromes or other associated conditions, efficacy of palate repair and unrepaired residual cleft or presence of fistula, status of velopharyngeal function and hearing status over time all of which eventually affect the ability to interact negatively. [4]

Considering the various multifactorial issues, a stereotypical interdisciplinary care is not always enough to deal with all the necessities of cleft palate management. Cleft affects various functions including that of the upper aero digestive tract like breathing, speech and on several occasions - even mastication. [2] It is an established fact that speech is altered in cleft children. The two milestones of speech which affect the outcomes are articulation, voice, and resonance. [5] Reasons like nasal obstruction, compromised hearing abilities and surgical fistulae have also been implicated in speech impairment. In our exhaustive review we came across a cross linguistic study which shows no significant difference in nasal resonance compared to normal children when the cases were operated at five months of age. [6] The cleft surgeon walks a fine thread in his attempt to balance speech versus skeletal growth. It is a race against time in deciding regarding the dilemma between late and early rehabilitation. On one hand late rehabilitation may lead to impaired speech whereas early rehabilitation may pose serious problems in the skeletal growth. And it is believed that this decision makes all the difference. [7]

Speech abnormalities in cleft patients

Resonance, velopharyngeal function, articulation perceived airflow, grimace, intelligibility, voice quality and hyper nasality are the assessed types of speech variables. [8]

The communication disorders most commonly associated with individuals with cleft palate are those related to velopharyngeal dysfunction or incomplete separation of the oral and nasal cavities. When this separation is disturbed, a variety of alterations in speech can occur, including hyper-nasality, mixed resonance, weak pressure consonants, and compensatory articulation patterns. [9] There is a strong relationship between palatal clefting and the

presence of hyper-nasality. [10] The presence of hyper-nasality in a speaker is called as 'cleft palate speech'. A cephalometric study analysing various factors of cleft palate speech to find the relationship between hyper nasality and hypopharyngeal space concludes the distance between posterior pharyngeal wall or adenoid tissue and posterior edge of hard palate as the main factors having significant role in speech pathology. Studies also suggest that thickness of the posterior pharyngeal wall is not a very significant factor in determining velopharyngeal function. [11]

For a long time, it was believed that the velopharynx is a simple two valve system with two positions: open or closed. Later, research and clinical observations have shown that the velo pharynx is a complex three-dimensional valve with a variety of shapes and structures. [7] Velopharyngeal dysfunction can be caused due to lack of tissue (velopharyngeal insufficiency) or lack of adequate movement (velopharyngeal incompetence) of the walls. [10]

Children with cleft palate have a high occurrence of voice problems such as hoarseness, breathlessness, low intensity, and pitch variations. It has been shown that children with cleft palate also demonstrate a high occurrence of laryngeal pathology including vocal nodules, vocal fold oedema or inflammatory reactions. [12] The data supports our assumption that the phonation disorders are more frequent in children with cleft palate than in normal children. [13]

The role of speech pathologist

By taking all the above mentioned details into account, a speech pathologist is an important partner to a surgeon in assessing the loco-pharyngeal insufficiency and thereby planning the treatment accordingly. [14]

According to a study conducted to investigate the validity and reliability of multiple listeners, hyper nasality and audible nasal emission in children with repaired cleft palate concludes as parents who were trained by a speech pathologist had constant or better reliability rate resonance and audible nasal emission, turbulence in speech of children with operated cleft palate and lip. [15] It is proven that training given to a parent of cleft child by a speech pathologist helps improve or stimulate the growth in vocabulary and thereby to expand the child's phonetic repertoire in day to day life. [16] In rare cases speech impairment maybe complicated by the presence of a common multianomaly syndrome such as Velocardiofacial Syndrome. This syndrome is signified by characteristic facies, conotruncal heart anomalies, palatal clefting (overt, submucous,

or occult submucous), learning disabilities, and behavioral disorders. Overlying syndromic conditions make treatment trickier than it already is. [17]

In such situations the role of sophisticated diagnostic modalities like Video-endo-naso-scopy and auditory perceptual assessments is stressed upon as an aid in correct diagnosis and early intervention. [18] Thus, it comes as no surprise when surgeons believe that first assessment of communication skills should begin in early infancy much before the child begins to speak, focusing mainly on the emerging sound production. [19]

The myriad list of problems in a cleft patient can be addressed by the collaboration of a speech pathologist and the surgeon in meeting the developmental milestones in the best way possible. [20]

Conclusion

The presence of a cleft palate presents as a social stigma even in today's era. It also negatively impacts an individual's ability to communicate effectively and may therefore cause significant social, emotional, and educational problems.

A surgeon in association with a speech pathologist can definitely bring about a positive change in the final speech outcome post surgically. Thus, the evaluation as well as the management of cleft palate is a critical part of comprehensive and effective cleft care. In the interdisciplinary team, the role of a speech pathologist cannot be stressed upon enough, especially in assessing and molding the surgical outcome.

On a concluding note the success arm is based on proper evaluation and decision making regarding the best method or technique for a particular type of defect which can be made with the feedback of a speech pathologist. It can be noteworthy in achieving ultimate aesthetic and functional harmony.

“CLEFT IS NOT A DISEASE” - IT IS UP TO US TO NOT TREAT IT LIKE ONE.

References

1. American Cleft Palate-Craniofacial Association. Revised Edition. Chapel Hill: American Cleft Palate-Craniofacial Association; 2004. Oct, Parameters for evaluation and treatment of patients with cleft lip/palate or other craniofacial anomalies.
2. Martin MC, D'Antonio L. The role of the speech pathologist in the care of the patient with cleft palate. In Booth PW, Schendel S, Hausamen J (eds). *Maxillofacial Surgery*, 2nd edn. St. Louis: Elsevier, pp. 1092–1101, 2005
3. Lohmander A, Olsson M : Methodology for Perceptual Assessment of Speech in Patients With Cleft Palate: A Critical Review of the Literature. *The Cleft Palate-Craniofacial Journal*. 2004;41(1):65-70
4. Chapman K. Is presurgery and early postsurgery performance related to speech and language outcomes at 3 years of age for children with cleft palate? *Clin Linguist Phon* 18(4-5):235–257, 2004.
5. Bessell A, Sell D, Whiting P, Roulstone S, Albery L, Persson M, Verhoeven A, Burke M, Ness A: Speech and Language Therapy Interventions for Children With Cleft Palate: A Systematic Review, *The Cleft Palate-Craniofacial Journal*. 2013;50(1):1-17.
6. Nancy J. Scherer, Linda L. D'Antonio, Holly McGahey, Early Intervention for Speech Impairment in Children With Cleft Palate, *The Cleft Palate-Craniofacial Journal*. 2008;45(1):18-31
7. Croft CB, Shprintzen RJ, Rakoff SJ. Patterns of velopharyngeal valving in normal and cleft palate subjects: A multi-view of videofluoroscopic and nasendoscopic study. *Laryngoscope* 91:265–271, 1981
8. Trost-Cardamone JE. Diagnosis of specific cleft palate speech error patterns for planning therapy of physical management needs. In: Bzoch KR, editor. *Communicative disorders related to cleft lip and palate*. Austin, TX: Pro-Ed; 1997.
9. Peterson-Falzone SJ, Hardin-Jones MA, Karnell MP. *Cleft palate speech*. Mosby: St Louis, MO; 2001
10. Trost-Cardamone JE. Coming to terms with VPI: A response to Loney and Bloem. *Cleft Palate J*. 1989;26:68–70
11. Lohmander A, Friede H, Lilja J: Long-Term, Longitudinal Follow-Up of Individuals With Unilateral Cleft Lip and Palate After the Gothenburg Primary Early Veloplasty and Delayed Hard Palate Closure Protocol: Speech Outcome, *The Cleft Palate-Craniofacial Journal*. 2012;49(6):657-67
12. Chapman KL, Hardin-Jones M, Schulte J, Halter KA. Vocal development of 9-month old babies with cleft palate. *J Speech Lang Hear Res*. 2001;44:1268–8

13. Lohmander-Agerskov A, Soderpalm E, Friede H, Lilja J. A comparison of babbling and speech at pre-speech level, 3, and 5 years of age in children with cleft lip and palate treated with delayed hard palate closure. *Folia PhoniatrLogop.* 1998;50:320–323
14. Bardach J, Morris HL, Jones G. The Iowa-Hamburg Project: late results of multidisciplinary management at the Iowa Cleft Palate Center. In Bardach J, Morris HL (eds). *Multidisciplinary Management of Cleft Lip and Palate.* Philadelphia: W.B. Saunders Co., 1990, pp. 98–112
15. Casby MW: Otitis media and language development: A meta-analysis. *American J Speech-Lang Pathol* 10:65–80, 2001
16. Folkins JW. Velopharyngeal nomenclature: Incompetency, inadequacy, insufficiency, and dysfunction. *Cleft Palate J.* 1988;25:413–6
17. Henningsson G, Kuehn DP, Sell D, Sweeney T, Trost-Cardamone JE, Whitehill TL. Universal parameters for reporting speech outcomes in individuals with cleft palate. *Cleft Palate Craniofac J.* 2008;45:1
18. D'Antonio L, Scherer NJ, Miller L, et al. Analysis of speech characteristics in children with velocardiofacial syndrome (VCFS) and children with phenotype overlap without VCFS. *Cleft Palate-Craniofac J* 38(5):455–467, 2001.
19. Nagarajan R, Savitha V.H., Subramaniyan B: Communication disorders in individuals with cleft lip and palate: An overview, *Indian J Plast Surg.* 2009;42(Suppl):137–143.
20. McWilliams BJ, Lavorato AS, Lavorato CD. Vocal cord abnormalities in children with velopharyngeal valving problems. *Laryngoscope* 83:1745–1753, 1973.

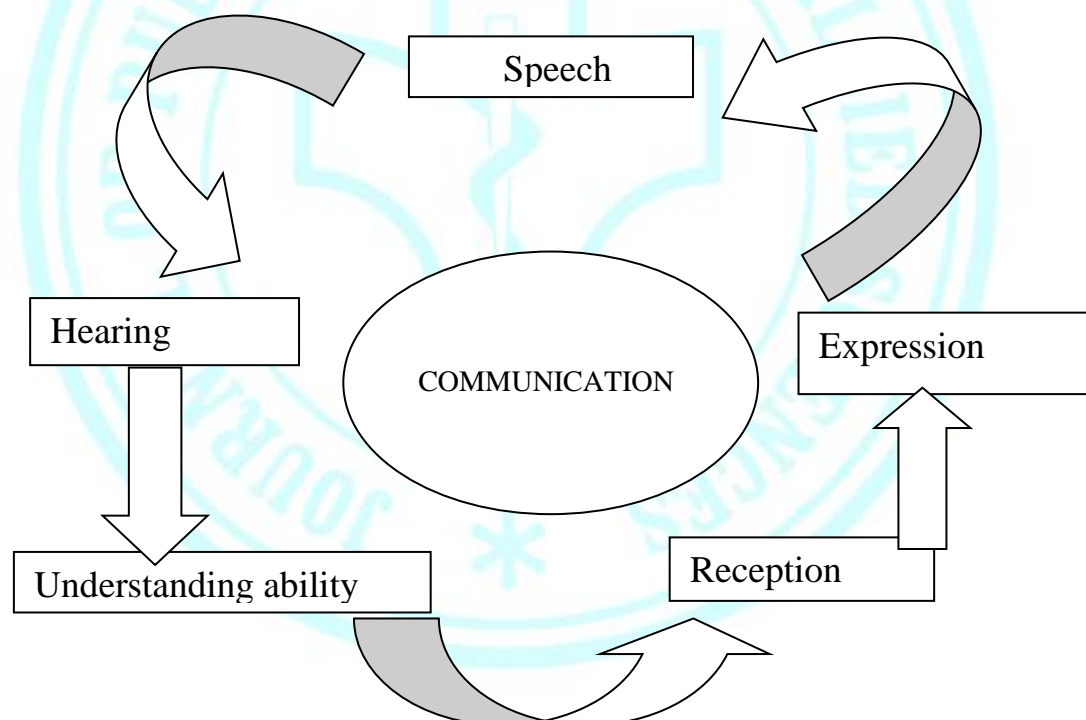


Table 1. This flowchart shows the cycle of complete speech.

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Review Article

An Insight of Astronaut's Dental Emergencies, Training and Preparations

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Abstract

It has been observed that extreme environment plays a significant effect on the oral cavity. One of the such effects has been observed in space mission where astronauts have encountered certain dental emergencies and later has been seen that astronauts and crew members have had serious effects on the health of the oral cavity. This article highlights various dental emergencies experienced during space mission and necessary steps that are been taken to avoid these emergencies. Goal of this article is to provide glimpse of the preparation, training and precaution that are taken by the astronauts in order to avoid any complications during the space mission.

Key words: Space dental emergencies, Astronaut dental training, Space dentistry.

Introduction

Space mission, whether of a short or long duration often has many medical effects on the astronaut. The reason being the simple fact that the astronauts in the space encounter vigorous working atmosphere requiring a great deal of endurance and efforts. Medical science has documented some of the complexities seen in astronauts as having affected their human physiology such as bone loss, cardiovascular diseases, strokes, altered trait, muscle hypotrophy, vision impairment due to intracranial pressure (VIIP) syndrome. [1,2,3,4] But recently some of the studies and statistics have shown that dental emergencies can be a huge hindrance for the astronaut during the space mission. [5,6] It has also been seen that apart from the medical complexities, unfavourable environment of the space is a pathophysiological factor for the many oral health related issues

confronted by the astronauts and also to the space pilot.

Documented studies

Due to limitation of various factors only few studies have been documented in relation to the effect on the oral health deterioration experienced by the short or long exposure to the space environment. [4]

These studies have concluded some of the very important aspects which have been challenged on the oral health of the astronauts. [3,4,10,11]

According to the studies, there has been prominent increase amount of dental plaque, calculus and inflammation of gingiva. [10,11] It has also been observed that a significant increase in number as well as activity of oral microorganism in the oral cavity of the astronauts. [10, 11]

It was found that there is a decrease flow rate of saliva which has resulted in significant increase in periodontal diseases and cavities. Decrease in saliva flow has many adverse effects on the oral cavity and is a serious issue which cannot be neglected.

Other problems that are documented in the studies include extreme facial and temporomandibular joint pain, altered taste sensation and delayed wound healing has also been a matter of concern in the past.

Possible dental emergencies experienced during space mission

It crucial to acknowledge the types of dental emergencies that may be present during a space mission and its possible consequences. Some of dental problems which can be addressed by the astronauts are as follows:

- 1) Barodontalgia: Pain in Tooth due to alteration in atmospheric pressure
- 2) Generalized periodontitis and gingival inflammation
- 3) Bone fracture and delayed wound healing
- 4) Decrease saliva flow, xerostomia and stones in the salivary gland ducts
- 5) Dental caries
- 6) Severe facial and jaw pain
- 7) Numbness of tooth and soft oral tissues
- 8) Headache and sinusitis

Preparation for the dental emergencies

It's very crucial that all the crew members should be aware of the possible emergencies and above all, they should be trained in tackling the situation and understanding the necessary precautions that need to be implemented. Preparation for these emergencies needs to be begun well before the departure for the space mission.

Pre-flight preparations

It's very essential to conduct a thorough oral as well as extra oral examination. All the necessary primary care for oral hygiene needs to be given utmost importance. It's very crucial to carry out all the obligatory dental treatments before-hand and avoid any maleficence concerned with oral cavity.

Astronaut and crew mandatory dental training

All the crew members including astronaut are required to go through dental training. Every member is taught and trained in emergency dental treatments like oral diagnosis, pain control, bleeding control, filling and even extraction if required during the mission. Training also includes deep understanding of all the equipment and infuses with unfamiliar techniques.

Apart from this, many easy documented pictures and step by step protocols in terms of graphs and diagrams are given to the crew members so that during emergency situation they can read and follow every instruction written for various dental procedures.

Conclusion

A consolidated and sophisticated knowledge of possible dental treatments and emergency procedure is important for the space mission crew and the astronauts. A well designed training and treatment protocol needs to be framed in order to avoid these discomforts during the space mission. A well oriented training session and proper prior examination are of paramount importance for the astronauts. All the above-mentioned feedbacks and suggestions need to be implemented to accomplish the success of space mission and proper measures are required for the overall safety of the astronauts.

References

1. [THE 6TH INTERNATIONAL CONGRESS "MEDICINE IN SPACE AND EXTREME ENVIRONMENTS"]. *Aviakosm Ekolog Med.* 2015 ;49(3):61-7.
2. Clă@Ment GR, Bukley AP, Paloski WH. Artificial gravity as a countermeasure for mitigating physiological deconditioning during long-duration space missions. *Front Syst Neurosci Frontiers in Systems Neuroscience.* 2015;9.
3. Rai B, Kaur J, Foing BH. Evaluation by an Aeronautic Dentist on the Adverse Effects of a Six-Week Period of Microgravity on the Oral Cavity. *International Journal of Dentistry.* 2011;2011:1–5.
4. Rai B, Kaur J. The history and importance of aeronautic dentistry. *J Oral Sci Journal of Oral Science.* 2011;53(2):143–6.
5. Brown LR, Fromme WJ, Handler SF, Wheatcroft MG, Johnston DA. Effect of Skylab missions on clinical and microbiologic aspects of oral health. *The Journal of the American Dental Association.* 1976;93(2):357–63.
6. Stewart LH, Trunkey D, Rebagliati GS. Emergency medicine in space. *The Journal of Emergency Medicine.* 2007;32(1):45–54.
7. Taylor GR, Kropp KD, Henney MR, Ekblad SS, Baky AA, Groves TO, Molina TC, Decelle JG, Carmichael CF, Gehring NJ, Young EL, Shannon IL, Frome WJ, FunderburkNR. Microbial Exchange: Experiment AR-002. *ApolloSoyuz Test Project: Summary Science Report Vol I. Scientific and Technical Information Office, National Aeronautics and Space Administration.* 1977; 237-255
8. Taylor GR. Recovery of medically important microorganisms from Apollo astronauts. *Aerospace Med.* 1974; 45: 824-828
9. Rai B, Kaur J. Periodontal status, salivary immunoglobulin, and microbial counts after short exposure to an isolated environment. *J Oral Sci Journal of Oral Science.* 2013;55(2):139–43.
10. Rai B, Kaur J, Foing BH. Wound Healing and Mucosal Immunity During Short Mars Analog Environment Mission: Salivary Biomarkers and Its Clinical Implications. *The Eurasian Journal of Medicine EAJM.* 2012Jan;44(2):63–7.
11. Rai B. Dentist on Mars: Personal View. *J Aeronaut Aerospace Eng Journal of Aeronautics & Aerospace Engineering.* 2012;01(02)

Child Sexual Abuse in India

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Introduction

Child sexual abuse, as defined by the World Health Organization, is the involvement of a child in sexual activity that he or she does not fully comprehend, is unable to give informed consent to, or that violates the laws of the society. [1]

Prevalence

India is home to almost 19% of the world's children; nearly 40% of its population and 69% of Indian children are victims of different forms of child abuse. [2] According to a study conducted by the Ministry of Women and Child Development [1], almost 53% of boys and 47% of girls – both aged between 5 and 18 years - across Indian states have faced at least one form of sexual abuse. The abuse ranges from being forced to touch private parts of their body and exposing a child to pornographic material to penetrative assault and being shot for pornographic material. The study also attacked the popular belief that boys are not vulnerable to sexual abuse and that all perpetrators of abuse are male. [1] Child trafficking, child marriage, child prostitution and child pornography are major contributors to child sexual abuse in India. [3] India has the largest number of child brides in the world, with 47% girls married under the legal age of 18 as per UNFPA. [3] According to a study conducted by Child Rights & You, an NGO, [4]:

1. 8,945 children go missing in India every year.
2. 500,000 children are estimated to be forced into the sex trade every year
3. Approximately 5.3 million child commercial sex workers are between the ages of 5 and 18 years
4. Children constitute 40% of the total population of commercial sex workers.

Evidently, children in unsafe environments - child labourers, street children, out of school children, impoverished children - are relatively more

vulnerable to sexual abuse as compared to children in homes and other trusted environments. However, children in comfortable and trusted spaces are not safe either. As is evident from Figure 1, at least 41% of sexual abuse is perpetrated by a trusted member of the family. [1,5]

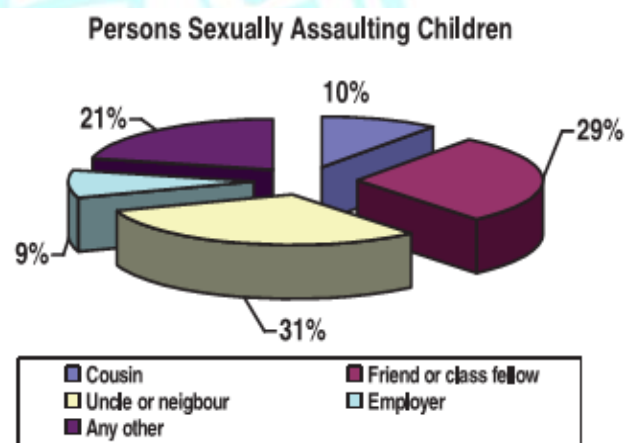


Figure 1

As the numbers suggest, every child in India, irrespective of socio-economic class, religion, caste and gender is at the risk of facing at least one form of sexual abuse. 53% boys and 47% girls being sexually abused [1] indicates that almost every second child is being sexually abused.

POCSO Act, 2012

The Protection of Children from Sexual Offences' Act had been passed by the Parliament in the year 2012 as a consequence of India acceding to the Convention on the Rights of the Child adopted by the General Assembly of the United Nations in 1992. [6]

The Act, the first of its kind in India, recognizes a wide gamut of probable perpetrators of child sexual abuse (including persons in a shared household &

custodians at institutional homes) and criminalizes the whole spectrum of sexually abusive actions against children (including attempt at abuse to types of penetration, involvement in pornography, sexual cyber-bullying and so on). The presumption

Besides, the awareness about the provision needs to substantially grow among parents, care takers and children before they can be duly protected by it. [10] A study conducted with 100 rural & urban parents from Jalandhar [2] showed that only 60%

Table 1

Percentage of children among different evidence groups reporting sexual assault		
	YES	NO
Child in family environment not going to school	4.04	95.96
Children in schools	2.90	97.10
Children at work (Shop, factory or other places)	8.70	91.30
Children on the streets	6.53	93.47
Children in institutional care	7.08	92.92
Total	5.69	94.31

of 'guilty of the accused until proven innocent' is also considered to be extraordinary. [7]

The Act has also prescribed procedures to report, record CSA cases & evidence and for running of Special Courts for speedy trial while protecting the child's Right to Confidentiality and Privacy. It provides the specific and tough punishments in accordance with the nature of sexual abuse. Imprisonment can vary from 3-10 years and beyond, depending on the severity of abuse. The Act is being mindful of the added trauma that the procedures could cause for the child and attempts to minimize it by outlining child-friendly procedures and ensuring rehabilitation for the child. [6]

It is undoubtedly commendable that the Act makes such a holistic attempt to sufficiently punish Child Sexual Abuse. Although it comes 20 years after India acceded to the Convention on the Rights of the Child in 1992, it is a firm step forward in empowering children. However, there are some pertinent issues identified in the scope of the Act that could hamper the effectiveness at Legislative, Judicial and Administrative levels [8] with legal, psychological social and medical implications. [9]

of urban parents and less than 40% of rural parents had a good knowledge about different forms of child abuse, sexual abuse being one among them.

The Central & State governments have been entrusted with the responsibility for spreading awareness about the POCSO Act through media. [11] However, so far, only a small but growing number of Non Government Organizations have been vigorously working on awareness among children and parents/caretakers resulting in a sharp rise in the number of rape cases being reported. [7]

In a brisk response to the legislation of the POCSO Act, the Ministry of Women & Child Development developed the 'Model Guidelines under Section 39 of the POCSO Act, 2012' which briefly outlines the principals, procedures, practices and for professionals working with providing legal, medical & psychological aid to children who have undergone & reported sexual abuse. The objective of this document is to foster better response mechanisms to promote the evolution of a multi-sectoral and multi-disciplinary approach to achieve the objectives of POCSO Act, 2012. [12]

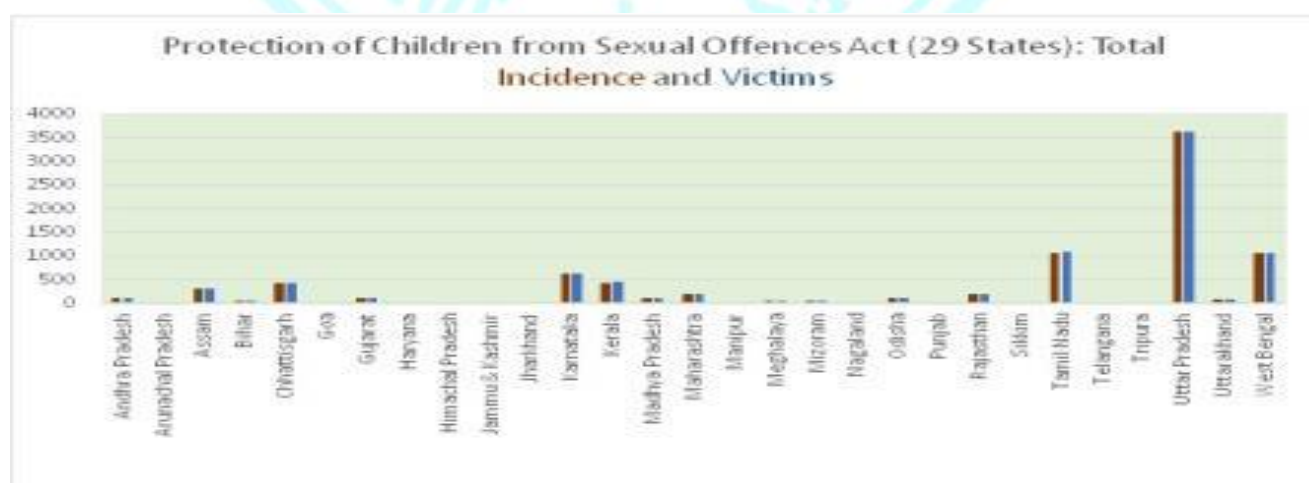


Figure 2

Reporting Abuse

A fundamental step in harnessing the efforts and provisions made by the government is the child being able to communicate the occurrence of the abuse to the immediate caretaker. Consider the data from Table 1 from a Study on Child Abuse in India. [1] This shows that an overall of 94.31% of sexual abuse of the most severe form – penetration – is not reported to any immediate caretaker. These numbers do not vary much among different forms of sexual abuse. [1] More than 48,000 child rape cases were recorded from 2001 to 2011 and India saw an increase of 336% of child rape cases from 2001 (2,113 cases) to 2011 (7,112 cases). [2] A comparison of these statistics shows that an increased number of reported cases is an indicator of a much more rapid increase in the incidence of child sexual abuse besides a gradual increase in awareness. The National Crime Records Bureau Report of 2014 indicates only a marginal increase in the reporting of child sexual abuse cases in different states (Figure 2). [8]

The lack of communication in terms of awareness, and dialogue within the family unit serves at a severe bottleneck in reporting child sexual abuse. Child abuse is a community problem and unless the community is aware of the issue, the extent of the problem and of their role in stopping and preventing child abuse, more and more children, families and communities will suffer. [2] Children also refrain from reporting abuse out of the fear of retribution from the abusers for the child reporting the incident. [5]

Method

Considering the staggering statistics of abuse and reporting, a minor study was performed. The objective of the study was threefold. Firstly, to observe how many young adults would be open to respond or converse on the issue of child sexual abuse. Secondly, to see how many young adults would be willing to share their childhood experiences, if any, of sexual abuse. Thirdly, to understand the impact that the unfortunate experience has had on them.

A post was shared with a primary network comprising of young adults and adults on a social networking website. The post shared a childhood experience of sexual abuse and sought others to share any experiences that they might have had as children.

Fourteen responses were received from across the country from primary and secondary networks. The respondents chose to share their CSA experiences to varying extents of details through varied modes of communication such as anonymous electronic mail, telephonic conversation and in-person meetings. Each of them shared at least one experience related to CSA. The information was collected through a personal interview. With their

consent, the same is being used here by replacing their names with pseudonyms to protect their identity

Results

Age: The respondents were subjected to CSA between ages 5 to 16.

Gender: 13 of the 14 respondents were female.

Perpetrators: 19 CSA experiences were shared by the 14 respondents. Out of the 18 perpetrators, only 2 were strangers. Consistent with the research done by Kacker *et al* [1], most of the perpetrators were persons trusted by the child and/or the family – neighbours, members of the family and teachers.

Reporting:

4 of the respondents could directly or indirectly share their 5 experiences with their parents and were met with a support by the latter.

2 of the experiences were shared with a parent only years after their occurrence and were met with understanding and support.

1 experience of a respondent, RK, was immediately reported to the parents whose response to the incident was unsupportive.

2 of the experiences were shared with one of the parents many years later only to be blamed by the parent for the occurrence and showing no anger towards the perpetrator, whatsoever.

9 of the CSA experiences that 7 of the respondents underwent were never communicated to their parents. In fact, 6 of the 14 respondents were sharing their experience for the first time with anyone. Consistent this is the finding that 72.1% of abused children never share their story with anyone. [1]

Awareness & Communication: None of the respondents were made aware – by their parents or other caregivers - of child abuse and personal safety as children.

Impact of abuse: All 14 respondents shared at least one adverse long term and short term impact on their psychological well-being as a result of the abuse.

Discussion

Impact of reporting

In 5 instances, the parents who were immediately notified of the abuse showed a positive response. They ensured that the children felt safe and either distanced the perpetrator from the family or ensured that the child does not feel threatened by the perpetrator's inevitable presence. AK, a female who could confidently report both her experiences to her parents, shares that it was probably her mother's experience of sexual abuse as a child that sensitized her sufficiently to protect AK from the perpetrator and the impact.

RK was a 15 years old female who was harassed by a stranger in the neighbourhood. Her family was

immediately alerted by her visible wounds. While they were prompt with the medical aid required, she was blamed for the occurrence. She was shamed for the clothes and behaviour – although these were no different from her regular clothes and behaviour. As a result, RK shares that she has no respect for member of her family who mocked and blamed her. 10 years later she still feels isolated and let down by her family and can never trust them.

Evidently, the nature of the parent/caretaker's response, whether they were informed immediately after the abuse or years later, goes a long way in impacting the survivor. Along with RK, RP and others who received unsupportive responses for their care-givers share that this can be as damaging as the abuse itself as it sends out the message the abuse was not a mistake of the perpetrator. It reinforces the sense of powerlessness that the abuse had established [13]. A healthy response, on the other hand, can mitigate the impact of the abuse to a great extent. AK shares that although she has trust issues when it comes to men, her parents' prompt & positive response strengthen her relationship with them and made her a more confident and secure person.

Silence

For MA and AT, this study was the only opportunity they perceived to be one where they can share with someone who would not undermine the abuse or its impact. For MK and SS sharing their experience with anyone was unthinkable until they saw the post put up by a trust-worthy member of their social network. SM and SP on the other hand did not want to share their experience at the cost of their parent's comfort and hence dealt with it on their own. This snapshot study in its limited sample shows a variety of reasons due to which the abuse goes unreported.

Unawareness

In the widespread scenario of limited awareness among children, particularly those aged below 8, there is only an intuition that alerts them that something is not right. However, this may or may not enable them to stop the abuse. Although TS, MK, AT, SS, MA and RP – all girls aged under 7 at the time of their first experience of sexual abuse - were not comfortable with what was happening, they did not know it was wrong and was to communicate to their parents. Their feelings about the occurrence were ambiguous and even overshadowed by the trust they had in the perpetrators. This is probably because we are taught to trust and respect our elders regardless of what they are doing. While this is a cultural practices and a strong family values, it is an attitude that violates the rights of the child. [14]

In fact, although SS and AT remember feeling 'dirty' after their repeated instances of abuse, they never happened to protest against it. Once TS and the others realised - through media or puberty talks by mothers around the age of 12 – the real impact of CSA set in. RT was in denial for a long time and told herself that she her story of abuse is a product of her imagination. She hated herself through her childhood and adolescence for degrading the 'innocent' perpetrator so such an extent in her imagination.

Both the issues – silence and unawareness - lead us to the stigma ingrained in our parenting practices which prevents conversation about abuse between children, parents and caregivers. Child rearing practices reflect social norms and very often adverse traditions are passed from one generation to the next [10].

The stigma attached to speaking about the body, sex, abuse and other such topics reduces children to quietly suffering from their abuse in isolation. The stigma insensitively attached to the abused further inhibits parents from sufficiently supporting their abused children. The stigma therefore reproduces and sustains itself sufficiently to sustain & protect Child sexual abuse - India's deepest most jointly held secret. [15]

There is a pressing need to understand social norms and traditions and their effect on children and their right to safety; to condemn harmful practices and to support those that are positively protective. In order to progress towards this, a major attitudinal change in civil society is called for. [14]

Psychological Impact

Child abuse is like a virus that attacks the host organism and alters it physically and psychologically. [2] The nature and extent of the effects of CSA are dependent on multiple factors - the child's age at the time of abuse, use of force, penetration, power dynamics and relationship with the perpetrator. [15].

A breakdown of trust followed by disgust & fear for the perpetrator are the most common reactions to CSA. [12] It is excruciating to come to terms with the fact that someone they trust can be capable of abusing them. The sense of powerlessness is immense when the child realises that the perpetrator is more powerful in the social/family structure. Once RP realised at the age of 8 that her cousin has been abusing her for the last 2 years, she decided against telling her parents whom she perceived as less powerful in the family than the cousin's parents. For a child to accept that scale of powerlessness can be demeaning and detrimental to her self-esteem.

If unresolved, the guilt, anger and negativity caused by the abuse can become an intrinsic part of one's self-image in the long run. The long term effects of CSA are seen as mental health & hygiene

problems. Many children subjected to sexual abuse develop substance abuse problems. The substance can include drugs and alcohol which reduced [13]. This could be one of the factors leading RT to indulge in undue amounts of tobacco and alcohol even at times of bad health.

Depression is another common problem seen among adolescents who were abused earlier. Constantly feeling powerless leads to learned helplessness wherein the child grows up telling him/herself that he/she has little control over their lives and circumstances. [12] SL and AS suffered from depression that went undiagnosed for a long time. They also suffered from associated symptoms of inflicting themselves with pain by cutting themselves to provide an outlet for the psychological pain they were undergoing.

SL also shares that following the abuse, she had suffered from Obsessive Compulsive Disorder that took her a while to control and regulate.

Adults, who grew up with the effects of CSA, may also develop traumatic sexualisation. [12] This means that they may develop sexual tendencies and desires which may seem inappropriate. Many are confused about their sexual orientation because of their earlier experiences. RK shared that her homosexual orientation might be an outcome of her being abused. MA had the courage to share that she attempted to abuse a younger cousin to see what it felt like to be the perpetrator. Virani [15] states that many abused girl children grow up to attempt at being the perpetrator in order to settle the power equation involved in the abuse. AT, under-confident at that time, admitted to indulging in sexual relationship with a partner at the tender age of 17, despite being aware that she was not prepared for it lest she be outcast from her friend circle for being the only one who is sexually inactive. SL also confessed to being unable to maintain boundaries in relationships, indulging in unwarranted sexual activity.

Struggling their way up to a healthy self-esteem [12] is a feature that most of the respondents spoke of. All the factors leading up to a healthy self-esteem sometimes don't naturally seem to outweigh the humiliation caused by a trusted adult, particularly in the eyes of the survivor. It is therefore a conscious effort that they have to put in besides allowing healthy support systems (most often the friends of the survivors) to see that the humiliation is not seen as a discredit but as an unfortunate incident.

As the long term effects imply, CSA initiates a self-perpetuating phenomenon through unhealthy relationships with oneself and with others. [15] As is evident, CSA is a physical violation that develops into a growing emotional violation if it goes unchecked.

Suggestions

To summarize, there broadly seem to be two sides to the coin of sexual abuse. On one side is the legislation of the POCSO Act, 2012 and its related Model Guidelines under Section 39 of the POCSO Act, 2012'. [12] On the same side, there are a growing number of NGOs working on awareness and resources to destigmatize CSA and make it a part of our everyday discourse.

Providing a huge impetus for this are mainstream Hindi commercial cinema such a Highway (2014) that compel the Indian audience to acknowledge the presence of CSA at the very least and noted celebrities like Kalki Koechlin who are encouraging people to converse about the issue by sharing their experience. Besides, to strengthen the discourse are also mainstream movies such as Balak Palak (2013) and Bollywood Talkies (2014) that talk about gender roles and stereotyping in the context of childhood & adolescence. These efforts are invaluable and represent the dawn of a movement to eradicate child sexual abuse not just by treating it but also endeavouring to prevent it.

On the other side of the same coin are our traditional values & culture that compel us to base our respect on age – thus, disrespecting children and decorating a multitude of abusers with dignity. Our societal norms do not provide scope for the conversation around a monumental and sensitive issue such as child sexual abuse, sexual education and sensitization and so on. Even our schools, being an offshoot of the same system, oversimplify sex education to a mere biological process and shun the more crucial aspects on rights, responsibilities, early awareness and so on.

Re-conceptualise Child sexual abuse

The feminist approach views rape as violation that is motivated not simply by lust but by aggression & the desire to dominate & control. [13] Understanding how rape harms the raped requires analysing it as an individual as well as at an institutional level – a structured social practice with implicit and explicit rules, positions and roles. According to Dworkin [13], rape is not committed by psychopaths or deviants of the social norms but by its exemplars who view oppressing as an entitlement.

Extending this view to Child Sexual Abuse and imbibing it will help the society view child sexual abuse not as an external evil that we are preventing from entering our system but as an internal parasite feeding on the flaws in our societal institutions. Hopefully the dire need of cultural shift in parenting practices and other such institutions [14] will be more effective under this view.

Awareness & Child empowerment

Interventions such as school-based programmes to prevent child sexual abuse have been incorporated

into the regular school curriculum in several countries equip children to recognize threatening situations, to protect themselves against abuse. [16] Considering that 41% of sexual abusers are trusted members in the child's immediate environment [1], children also need to be educated about abuse not only from strangers but from their parents, family members & teachers. [5]

The traditional family structure in India makes the children highly dependent on their parents and expects them to play submissive and obedient roles towards their elders. [17] The Indian view holds the children as parental property, subject to discipline as the parents find appropriate. [18] This is one of the main obstacles to reporting any form of abuse, particularly those that occur at home. [11]

The concept of a 'caring community' as children's right, conceived by Dr. Eric Ram argues that every service, infrastructure facility, person and institution touching a child's daily life has the potential to be a 'caring community' for children. It is an issue of attitude, of not just giving care to the child, but caring about what happens to a child, and thus honouring the ethics that should guide any dealings with any child. [14]

Re-positioning the status of the child in the family and society as an equal respected member with rights will help children see themselves as empowered individuals. Decreased abuse and increased reporting may just be one of the many advantages of this phenomenon. Building on the efforts of organizations to improve awareness among parents and children, families and schools need to approach the issue of child abuse with the reconceptualised view and with the objective of not just primary prevention [16] but also uprooting it.

It is unacceptable that in spite of the seriousness of the problem in terms of numbers and effects, the acknowledgement of CSA has met with great resistance world over. [15] The society is a bearer of the invisible but enormous shame that child sexual abuse is - the troubled childhood and potentially unhealthy development into adulthood, of more than half of our children. Ultimately, only adults who live in Indian homes can stop it. [15]

The reconceptualised view of child sexual abuse and the empowerment of children through awareness and societal re-positioning may be the route to sustainable eradication of Child Sexual Abuse.

References

1. Ministry of Women & Child Development. Study on Child Abuse: India 2007. New Delhi: Government of India; 2007
2. Singh J, Kaur B. A Comparative Study to assess the knowledge regarding Child Abuse among parents residing in selected rural and urban areas of Jalandhar, Punjab.

- International Journal of Development Research. 2015; 5(11):6001-6006.
3. Bajpai A. Child rights in India: Law, politics & practice. New Delhi: Oxford University Press; 2003
4. The sounds of silence: Child sexual abuse in India | Analysis | Children [Internet]. Infochangeindia.org. 2016 [cited 22 September 2013]. Available from: <http://infochangeindia.org/children/analysis/the-sounds-of-silence-child-sexual-abuse-in-india.html>
5. Tshabalala T, Khosa M. Awareness of Various Forms of Child Abuse: Pupil's Attributions - A case study of Gomadoda cluster. Asian Journal of Social Sciences & Management Sciences. 2014; :23-28.
6. The Ministry of Law & Justice. The Protection of Children from Sexual Offences Act of 2012. New Delhi: Government of India; 2012.
7. Belur J, Singh B. Child Sexual Abuse and the law in India: a commentary. Crime Science - a SpringerOpen Journal. 2015.
8. Kumar S. The Pocso Act: A Quick Review: SrishtiAgnihotri and Minakshi Das [Internet]. Kafila. 2015 [cited 7 August 2016]. Available from: <https://kafila.org/2015/12/10/the-pocso-act-a-quick-review-srishti-agnihotri-and-minakshi-das/>
9. Moirangthem S, Kumar N C, Math S B. Child Sexual Abuse: Issues & Concerns. Indian Journal of Medical Research. 2015;:1-3.
10. Seth R. Protection of Children From Abuse & Neglect in India. The 28th CMAAO General Assembly & 49th Council Meeting-Takemi Memorial Oration. Japan Medical Association Journal; 2013
11. [Internet] Childlineindia.org. 2016 [cited 10 August 2016]. Available from: <http://childlineindia.org.in/The-Protection-of-Children-from-Sexual-Offences-Act-2012>
12. Ministry of Women and Child Development. Model Guidelines under Section 39 of the POCSO Act, 2012. New Delhi: Government of India; 2013
13. Whisnant R. Feminist Perspectives on Rape [Internet]. Plato.stanford.edu. 2009 [cited 7 August 2016]. Available from: <http://plato.stanford.edu/entries/feminism-rape/>
14. Saini N. Child Abuse & Neglect in India: Time to act. The 28th CMAAO General Assembly & 49th Council Meeting-Takemi Memorial Oration. Japan Medical Association Journal; 2013

15. Virani P. Bitter Chocolate - Child Sexual Abuse in India. New Delhi: Penguin Books India Pvt. Ltd. 2000
16. World Health Organization. Child Abuse and Neglect by Parents and Other Caregivers. In Global campaign for Violence Prevention. p. 57 - 86.
17. [Internet] Childlineindia.org. 2016 [cited 10 August 2016]. Available from: <http://www.childlineindia.org.in/child-abuse-child-violence-india.htm>
18. UA S. Child abuse in India: an empirical report on perceptions. - PubMed - NCBI [Internet]. Ncbi.nlm.nih.gov. 2016 [cited 10 August 2016]. Available from: <http://www.ncbi.nlm.nih.gov/pubmed/148651>

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